HIV-1 Vif versus the APOBEC3 cytidine deaminases: an intracellular duel between pathogen and host restriction factors.

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Public Summary:
The Vif protein of HIV is essential for the effective propagation of this pathogenic retrovirus in vivo. Vif acts by preventing virion encapsidation of two potent antiviral factors, the APOBEC3G and APOBEC3F cytidine deaminases. Decreased encapsidation in part involves Vif-mediated recruitment of a ubiquitin E3 ligase complex that promotes polyubiquitylation and proteasome-mediated degradation of APOBEC3G/F. The resultant decline in intracellular levels of these enzymes leads to decreased encapsidation of APOBECG/F into budding virions. This review discusses recent advances in our understanding of the dynamic interplay of Vif with the antiviral APOBEC3 enzymes.

Scientific Abstract:
The Vif protein of HIV is essential for the effective propagation of this pathogenic retrovirus in vivo. Vif acts by preventing virion encapsidation of two potent antiviral factors, the APOBEC3G and APOBEC3F cytidine deaminases. Decreased encapsidation in part involves Vif-mediated recruitment of a ubiquitin E3 ligase complex that promotes polyubiquitylation and proteasome-mediated degradation of APOBEC3G/F. The resultant decline in intracellular levels of these enzymes leads to decreased encapsidation of APOBECG/F into budding virions. This review discusses recent advances in our understanding of the dynamic interplay of Vif with the antiviral APOBEC3 enzymes.